

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 16:04:11 ON 22 JUL 2008

=> fil .bec

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS,  
ESBIOBASE, BIOTECHNO, WPIDS' ENTERED AT 16:04:32 ON 22 JUL 2008  
ALL COPYRIGHTS AND RESTRICTIONS APPLY. SEE HELP USAGETERMS FOR DETAILS.

11 FILES IN THE FILE LIST

=> s catalase#

FILE 'MEDLINE'

L1 30094 CATALASE#

FILE 'SCISEARCH'

L2 24866 CATALASE#

FILE 'LIFESCI'

L3 8234 CATALASE#

FILE 'BIOTECHDS'

L4 1569 CATALASE#

FILE 'BIOSIS'

L5 40330 CATALASE#

FILE 'EMBASE'

L6 25131 CATALASE#

FILE 'HCAPLUS'

L7 51557 CATALASE#

FILE 'NTIS'

L8 240 CATALASE#

FILE 'ESBIOBASE'

L9 13053 CATALASE#

FILE 'BIOTECHNO'

L10 5716 CATALASE#

FILE 'WPIDS'

L11 2331 CATALASE#

TOTAL FOR ALL FILES

L12 203121 CATALASE#

=> s l12 and peroxisom?

FILE 'MEDLINE'

16590 PEROXISOM?

L13 1642 L1 AND PEROXISOM?

FILE 'SCISEARCH'

19200 PEROXISOM?

L14 1147 L2 AND PEROXISOM?

FILE 'LIFESCI'

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      4905 PEROXISOM?
L15      369 L3 AND PEROXISOM?

FILE 'BIOTECHDS'
      571 PEROXISOM?
L16      36 L4 AND PEROXISOM?

FILE 'BIOSIS'
      22461 PEROXISOM?
L17      2070 L5 AND PEROXISOM?

FILE 'EMBASE'
      20383 PEROXISOM?
L18      1423 L6 AND PEROXISOM?

FILE 'HCAPLUS'
      23987 PEROXISOM?
L19      2057 L7 AND PEROXISOM?

FILE 'NTIS'
      86 PEROXISOM?
L20      6 L8 AND PEROXISOM?

FILE 'ESBIOBASE'
      11023 PEROXISOM?
L21      533 L9 AND PEROXISOM?

FILE 'BIOTECHNO'
      4748 PEROXISOM?
L22      403 L10 AND PEROXISOM?

FILE 'WPIDS'
      1859 PEROXISOM?
L23      13 L11 AND PEROXISOM?

TOTAL FOR ALL FILES
L24      9699 L12 AND PEROXISOM?

=> s peroxisom?(10a)(target? or import? or transport?)
FILE 'MEDLINE'
      16590 PEROXISOM?
      433960 TARGET?
      1140741 IMPORT?
      366787 TRANSPORT?
L25      2117 PEROXISOM?(10A)(TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'SCISEARCH'
      19200 PEROXISOM?
      512182 TARGET?
      1289370 IMPORT?
      572388 TRANSPORT?
L26      2114 PEROXISOM?(10A)(TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'LIFESCI'
      4905 PEROXISOM?
      165131 TARGET?
      356183 IMPORT?
      96290 TRANSPORT?
L27      840 PEROXISOM?(10A)(TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'BIOTECHDS'
      571 PEROXISOM?

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41384 TARGET?  
22583 IMPORT?  
8626 TRANSPORT?  
L28 85 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'BIOSIS'  
22461 PEROXISOM?  
408829 TARGET?  
1093452 IMPORT?  
2957525 TRANSPORT?  
L29 2200 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'EMBASE'  
20383 PEROXISOM?  
419044 TARGET?  
1042057 IMPORT?  
373283 TRANSPORT?  
L30 1799 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'HCAPLUS'  
23987 PEROXISOM?  
603337 TARGET?  
1281085 IMPORT?  
897323 TRANSPORT?  
L31 2764 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'NTIS'  
86 PEROXISOM?  
70106 TARGET?  
156178 IMPORT?  
147745 TRANSPORT?  
L32 4 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'ESBIOBASE'  
11023 PEROXISOM?  
266486 TARGET?  
501177 IMPORT?  
277295 TRANSPORT?  
L33 3059 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'BIOTECHNO'  
4748 PEROXISOM?  
111737 TARGET?  
184414 IMPORT?  
85418 TRANSPORT?  
L34 871 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

FILE 'WPIDS'  
1859 PEROXISOM?  
228981 TARGET?  
33091 IMPORT?  
362254 TRANSPORT?  
L35 74 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

TOTAL FOR ALL FILES  
L36 15927 PEROXISOM?(10A) (TARGET? OR IMPORT? OR TRANSPORT?)

=> s 112(15a)l36  
FILE 'MEDLINE'  
L37 62 L1 (15A)L25

FILE 'SCISEARCH'  
L38 68 L2 (15A)L26

FILE 'LIFESCI'  
L39 37 L3 (15A)L27

FILE 'BIOTECHDS'  
L40 2 L4 (15A)L28

FILE 'BIOSIS'  
L41 74 L5 (15A)L29

FILE 'EMBASE'  
L42 53 L6 (15A)L30

FILE 'HCAPLUS'  
L43 85 L7 (15A)L31

FILE 'NTIS'  
L44 0 L8 (15A)L32

FILE 'ESBIOBASE'  
L45 48 L9 (15A)L33

FILE 'BIOTECHNO'  
L46 40 L10(15A)L34

FILE 'WPIDS'  
L47 1 L11(15A)L35

TOTAL FOR ALL FILES  
L48 470 L12(15A) L36

=> s l12(5a)(treat? or pharmaceutical?)

FILE 'MEDLINE'  
2724985 TREAT?  
104540 PHARMACEUTICAL?  
L49 741 L1 (5A)(TREAT? OR PHARMACEUTICAL?)

FILE 'SCISEARCH'  
2150934 TREAT?  
50361 PHARMACEUTICAL?  
L50 685 L2 (5A)(TREAT? OR PHARMACEUTICAL?)

FILE 'LIFESCI'  
414886 TREAT?  
8156 PHARMACEUTICAL?  
L51 245 L3 (5A)(TREAT? OR PHARMACEUTICAL?)

FILE 'BIOTECHDS'  
112805 TREAT?  
29256 PHARMACEUTICAL?  
L52 54 L4 (5A)(TREAT? OR PHARMACEUTICAL?)

FILE 'BIOSIS'  
2322340 TREAT?  
176047 PHARMACEUTICAL?  
L53 992 L5 (5A)(TREAT? OR PHARMACEUTICAL?)

FILE 'EMBASE'  
2576754 TREAT?  
80562 PHARMACEUTICAL?

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L54          648 L6 (5A) (TREAT? OR PHARMACEUTICAL?)

FILE 'HCAPLUS'
    3821839 TREAT?
    372979 PHARMACEUTICAL?
L55          1815 L7 (5A) (TREAT? OR PHARMACEUTICAL?)

FILE 'NTIS'
    129998 TREAT?
    2460 PHARMACEUTICAL?
L56          4 L8 (5A) (TREAT? OR PHARMACEUTICAL?)

FILE 'ESBIOBASE'
    755842 TREAT?
    29630 PHARMACEUTICAL?
L57          551 L9 (5A) (TREAT? OR PHARMACEUTICAL?)

FILE 'BIOTECHNO'
    280839 TREAT?
    7365 PHARMACEUTICAL?
L58          167 L10(5A) (TREAT? OR PHARMACEUTICAL?)

FILE 'WPIDS'
    1172942 TREAT?
    187208 PHARMACEUTICAL?
L59          123 L11(5A) (TREAT? OR PHARMACEUTICAL?)

TOTAL FOR ALL FILES
L60          6025 L12(5A) (TREAT? OR PHARMACEUTICAL?)

=> s 124 and 160
FILE 'MEDLINE'
L61          42 L13 AND L49

FILE 'SCISEARCH'
L62          29 L14 AND L50

FILE 'LIFESCI'
L63          11 L15 AND L51

FILE 'BIOTECHDS'
L64          2 L16 AND L52

FILE 'BIOSIS'
L65          49 L17 AND L53

FILE 'EMBASE'
L66          39 L18 AND L54

FILE 'HCAPLUS'
L67          58 L19 AND L55

FILE 'NTIS'
L68          0 L20 AND L56

FILE 'ESBIOBASE'
L69          19 L21 AND L57

FILE 'BIOTECHNO'
L70          15 L22 AND L58

FILE 'WPIDS'

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L71 2 L23 AND L59

TOTAL FOR ALL FILES

L72 266 L24 AND L60

=> s (l48 or l72) not 2003-2008/py

FILE 'MEDLINE'

3553100 2003-2008/PY

(20030000-20089999/PY)

L73 80 (L37 OR L61) NOT 2003-2008/PY

FILE 'SCISEARCH'

6660421 2003-2008/PY

(20030000-20089999/PY)

L74 66 (L38 OR L62) NOT 2003-2008/PY

FILE 'LIFESCI'

802010 2003-2008/PY

L75 35 (L39 OR L63) NOT 2003-2008/PY

FILE 'BIOTECHDS'

138709 2003-2008/PY

L76 2 (L40 OR L64) NOT 2003-2008/PY

FILE 'BIOSIS'

3117126 2003-2008/PY

L77 97 (L41 OR L65) NOT 2003-2008/PY

FILE 'EMBASE'

3098181 2003-2008/PY

L78 72 (L42 OR L66) NOT 2003-2008/PY

FILE 'HCAPLUS'

7038476 2003-2008/PY

L79 111 (L43 OR L67) NOT 2003-2008/PY

FILE 'NTIS'

91535 2003-2008/PY

L80 0 (L44 OR L68) NOT 2003-2008/PY

FILE 'ESBIOBASE'

1777854 2003-2008/PY

L81 45 (L45 OR L69) NOT 2003-2008/PY

FILE 'BIOTECHNO'

122467 2003-2008/PY

L82 51 (L46 OR L70) NOT 2003-2008/PY

FILE 'WPIDS'

5853897 2003-2008/PY

L83 1 (L47 OR L71) NOT 2003-2008/PY

TOTAL FOR ALL FILES

L84 560 (L48 OR L72) NOT 2003-2008/PY

=> dup rem l84

PROCESSING COMPLETED FOR L84

L85 162 DUP REM L84 (398 DUPLICATES REMOVED)

=> d tot

L85 ANSWER 1 OF 162 MEDLINE on STN

DUPLICATE 1

TI Peroxisome senescence in human fibroblasts.  
SO Molecular biology of the cell, (2002 Dec) Vol. 13, No. 12, pp. 4243-55.  
Journal code: 9201390. ISSN: 1059-1524.  
AU Legakis Julie E; Koepke Jay I; Jedeszko Chris; Barlasakar Ferdous; Terlecky  
Laura J; Edwards Holly J; Walton Paul A; Terlecky Stanley R  
AN 2002713774 MEDLINE

L85 ANSWER 2 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI A novel pex2 mutant: catalase-deficient but temperature-sensitive PTS1 and  
PTS2 import  
SO Biochemical and Biophysical Research Communications (2002), 293(5),  
1523-1529  
CODEN: BBRCA9; ISSN: 0006-291X  
AU Akiyama, Noriko; Ghaedi, Kamran; Fujiki, Yukio  
AN 2002:465334 HCAPLUS  
DN 137:229905

L85 ANSWER 3 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
STN  
TI Catalase associated proteins in peroxisomes.  
SO FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp. A1190. print.  
Meeting Info.: Annual Meeting of Professional Research Scientists on  
Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.  
CODEN: FAJOEC. ISSN: 0892-6638.  
AU Olcum, Tulin [Reprint author]; Donaldson, Robert P. [Reprint author]  
AN 2002:472283 BIOSIS

L85 ANSWER 4 OF 162 Elsevier BIOBASE COPYRIGHT 2008 Elsevier Science B.V.  
on STN  
AN 2002181206 ESBIIOBASE  
TI The role of peroxisomes in the integration of metabolism and evolutionary  
diversity of photosynthetic organisms  
AU Igamberdiev A.U.; Lea P.J.  
CS Dr. A.U. Igamberdiev, Plant Research Department, Riso National  
Laboratory, 4000 Roskilde, Denmark.  
E-mail: abir.igamberdiev@risoe.dk  
SO Phytochemistry, (14 AUG 2002), 60/7 (651-674), 237 reference(s)  
CODEN: PYTCAS ISSN: 0031-9422  
PUI S0031942202001796  
DT Journal; General Review  
CY United Kingdom  
LA English  
SL English

L85 ANSWER 5 OF 162 MEDLINE on STN DUPLICATE 2  
TI Distribution and characterization of peroxisomes in Arabidopsis by  
visualization with GFP: dynamic morphology and actin-dependent movement.  
SO Plant & cell physiology, (2002 Mar) Vol. 43, No. 3, pp. 331-41.  
Journal code: 9430925. ISSN: 0032-0781.  
AU Mano Shoji; Nakamori Chihiro; Hayashi Makoto; Kato Akira; Kondo Maki;  
Nishimura Mikio  
AN 2002184616 MEDLINE

L85 ANSWER 6 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on  
STN  
TI Targeting mechanism of plant catalase into  
peroxisomes  
SO PLANT AND CELL PHYSIOLOGY, (2002) Vol. 43, Supp. [S], pp. S188-S188.  
ISSN: 0032-0781.  
AU Kamigaki A (Reprint); Terauchi K; Mano S; Nito K; Hayashi M; Nishimura M;  
Esaka M  
AN 2002:325803 SCISEARCH

L85 ANSWER 7 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN  
 TI Effects of growth hormone and insulin-like growth factor-1 on hepatocyte antioxidative enzymes  
 SO EXPERIMENTAL BIOLOGY AND MEDICINE, (FEB 2002) Vol. 227, No. 2, pp. 94-104. ISSN: 1535-3702.  
 AU Brown-Borg H M (Reprint); Rakoczy S G; Romanick M A; Kennedy M A  
 AN 2002:89745 SCISEARCH

L85 ANSWER 8 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
 TI H2O2-release from intact peroxisomes: new insights into peroxisome-derived oxidative stress  
 SO Proceedings of [the] Biennial Meeting of the Society for Free Radical Research International, 11th, Paris, France, July 16-20, 2002 (2002), 35-43. Editor(s): Pasquier, Catherine. Publisher: Monduzzi Editore, Bologna, Italy.  
 CODEN: 69EZI4; ISBN: 88-323-2716-3  
 AU Fritz, R.; Voelkl, A.; Stremmel, W.; Mueller, S.  
 AN 2004:44178 HCAPLUS  
 DN 140:176839

L85 ANSWER 9 OF 162 MEDLINE on STN DUPLICATE 3  
 TI In situ measurements of the pH of mammalian peroxisomes using the fluorescent protein pHluorin.  
 SO The Journal of biological chemistry, (2001 Dec 28) Vol. 276, No. 52, pp. 48748-53. Electronic Publication: 2001-10-18.  
 Journal code: 2985121R. ISSN: 0021-9258.  
 AU Jankowski A; Kim J H; Collins R F; Daneman R; Walton P; Grinstein S  
 AN 2002003433 MEDLINE

L85 ANSWER 10 OF 162 MEDLINE on STN DUPLICATE 4  
 TI Peroxisomal catalase in the methylotrophic yeast Candida boidinii: transport efficiency and metabolic significance.  
 SO Journal of bacteriology, (2001 Nov) Vol. 183, No. 21, pp. 6372-83.  
 Journal code: 2985120R. ISSN: 0021-9193.  
 AU Horiguchi H; Yurimoto H; Goh T; Nakagawa T; Kato N; Sakai Y  
 AN 2001544704 MEDLINE

L85 ANSWER 11 OF 162 MEDLINE on STN DUPLICATE 5  
 TI Multiple catalase genes are differentially regulated in Aspergillus nidulans.  
 SO Journal of bacteriology, (2001 Feb) Vol. 183, No. 4, pp. 1434-40.  
 Journal code: 2985120R. ISSN: 0021-9193.  
 AU Kawasaki L; Aguirre J  
 AN 2001179122 MEDLINE

L85 ANSWER 12 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
 TI Temperature sensitive acyl-CoA oxidase import in group A peroxisome biogenesis disorders  
 SO Journal of Medical Genetics (2001), 38(12), 871-874  
 CODEN: JMDGAE; ISSN: 0022-2593  
 AU Imamura, Atsushi; Shimozawa, Nobuyuki; Suzuki, Yasuyuki; Zhang, Zhongyi; Tsukamoto, Toshiro; Orii, Tadao; Osumi, Takashi; Kondo, Naomi  
 AN 2002:50163 HCAPLUS  
 DN 137:18746

L85 ANSWER 13 OF 162 MEDLINE on STN DUPLICATE 6  
 TI Oxidative stress, microsomal and peroxisomal fatty acid oxidation in the liver of rats treated with acetone.  
 SO Comparative biochemistry and physiology. Toxicology & pharmacology : CBP,

(2001 Apr) Vol. 128, No. 4, pp. 503-9.  
Journal code: 100959500. ISSN: 1532-0456.

AU Orellana B M; Guajardo V; Araya J; Thieleman L; Rodrigo R  
AN 2001483739 MEDLINE

L85 ANSWER 14 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
STN

TI Antioxidant system within yeast peroxisome: Metabolic  
significance and peroxisomal transport of  
catalase and a novel peroxiredoxine Pmp20.

SO Cell Structure and Function, (October, 2001) Vol. 26, No. 5, pp. 473.  
print.

Meeting Info.: Fifty-fourth Annual Meeting of the Japan Society for Cell  
Biology. Gifu, Japan. May 20-June 01, 2001. Japan Society for Cell  
Biology.

CODEN: CSFUDY. ISSN: 0386-7196.

AU Sakai, Yasuyoshi [Reprint author]; Horiguchi, Hirofumi [Reprint author];  
Yurimoto, Hiroya [Reprint author]; Kato, Nobuo [Reprint author]

AN 2002:189894 BIOSIS

L85 ANSWER 15 OF 162 MEDLINE on STN DUPLICATE 7

TI Differential induction of peroxisomal populations in subcellular  
fractions of rat liver.

SO Biochimica et biophysica acta, (2001 Jan 12) Vol. 1544, No. 1-2, pp.  
358-69.

Journal code: 0217513. ISSN: 0006-3002.

AU Wilcke M; Alexson S E

AN 2001306718 MEDLINE

L85 ANSWER 16 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Temperature-Sensitive Phenotype of Chinese Hamster Ovary Cells Defective  
in PEX5 Gene

SO Biochemical and Biophysical Research Communications (2001), 288(2),  
321-327

CODEN: BBRC9; ISSN: 0006-291X

AU Ito, Ritsu; Huang, Yuan; Yao, Can; Shimozawa, Nobuyuki; Suzuki, Yasuyuki;  
Kondo, Naomi; Imanaka, Tsuneo; Usuda, Nobuteru; Ito, Masaki

AN 2001:759950 HCAPLUS

DN 136:115955

L85 ANSWER 17 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Pex12p of Saccharomyces cerevisiae is a component of a multi-protein  
complex essential for peroxisomal matrix protein import

SO European Journal of Cell Biology (2001), 80(4), 257-270

CODEN: EJCBDN; ISSN: 0171-9335

AU Albertini, Markus; Girzalsky, Wolfgang; Veenhuis, Marten; Kunau, Wolf-H.

AN 2001:460674 HCAPLUS

DN 136:2690

L85 ANSWER 18 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation  
on STN

TI Antioxidant system within yeast peroxisome: Metabolic  
significance and peroxisomal transport of  
catalase and a novel peroxiredoxine Pmp20.

SO YEAST, (AUG 2001) Vol. 18, Supp. [1], pp. S129-S129.  
ISSN: 0749-503X.

AU Sakai Y (Reprint); Horiguchi H; Yurimoto H; Kato N

AN 2001:674755 SCISEARCH

L85 ANSWER 19 OF 162 MEDLINE on STN DUPLICATE 8

TI Studies of early hepatocellular proliferation and peroxisomal  
proliferation in Wistar rats treated with herbicide diclofop.

SO Toxicology, (2001 Feb 14) Vol. 158, No. 3, pp. 119-26.  
Journal code: 0361055. ISSN: 0300-483X.

AU Palut D; Ludwicki J K; Kostka G; Kopec-Szlezak J; Wiadrowska B; Lembowicz K

AN 2001183146 MEDLINE

L85 ANSWER 20 OF 162 MEDLINE on STN DUPLICATE 9

TI Catalase-less peroxisomes. Implication in the milder forms of peroxisome biogenesis disorder.

SO The Journal of biological chemistry, (2000 Nov 24) Vol. 275, No. 47, pp. 37271-7.  
Journal code: 2985121R. ISSN: 0021-9258.

AU Fujiwara C; Imamura A; Hashiguchi N; Shimozawa N; Suzuki Y; Kondo N; Imanaka T; Tsukamoto T; Osumi T

AN 2001082757 MEDLINE

L85 ANSWER 21 OF 162 MEDLINE on STN DUPLICATE 10

TI Toxoplasma gondii catalase: are there peroxisomes in toxoplasma?.

SO Journal of cell science, (2000 Jul) Vol. 113 ( Pt 13), pp. 2409-19.  
Journal code: 0052457. ISSN: 0021-9533.

AU Ding M; Clayton C; Soldati D

AN 2000437355 MEDLINE

L85 ANSWER 22 OF 162 MEDLINE on STN DUPLICATE 11

TI Immunological detection of alkaline-diaminobenzidine-negative peroxisomes of the nematode *Caenorhabditis elegans* purification and unique pH optima of peroxisomal catalase.

SO European journal of biochemistry / FEBS, (2000 Mar) Vol. 267, No. 5, pp. 1307-12.  
Journal code: 0107600. ISSN: 0014-2956.

AU Togo S H; Maebuchi M; Yokota S; Bun-Ya M; Kawahara A; Kamiryo T

AN 2000156233 MEDLINE

L85 ANSWER 23 OF 162 MEDLINE on STN DUPLICATE 12

TI Targeting and subcellular localization of *Toxoplasma gondii* catalase. Identification of peroxisomes in an apicomplexan parasite.

SO The Journal of biological chemistry, (2000 Jan 14) Vol. 275, No. 2, pp. 1112-8.  
Journal code: 2985121R. ISSN: 0021-9258.

AU Kaasch A J; Joiner K A

AN 2000092881 MEDLINE

L85 ANSWER 24 OF 162 BIOTECHDS COPYRIGHT 2008 THOMSON REUTERS on STN

TI Stabilization of entrapped catalase using photo-crosslinked resin gel for use in waste-water containing hydrogen peroxide; enzyme stabilization using Tween-80 surfactant prior to immobilization on photo-crosslinked resin gel support

SO J.Chem.Technol.Biotechnol.; (2000) 75, 11, 1026-30  
CODEN: JCTBDC ISSN: 0268-2575

AU Iida T; Muruyama D; Fukunaga K

AN 2000-14404 BIOTECHDS

L85 ANSWER 25 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

TI A Missense Mutation in the RING Finger Motif of PEX2 Protein Disturbs the Import of Peroxisome Targeting Signal 1 (PTS1)-Containing Protein but Not the PTS2-Containing Protein

SO Biochemical and Biophysical Research Communications (2000), 270(3), 717-721  
CODEN: BBRCA9; ISSN: 0006-291X

AU Huang, Yuan; Ito, Ritsu; Miura, Satoshi; Hashimoto, Takashi; Ito, Masaki

AN 2000:253750 HCAPLUS

DN 133:41022

L85 ANSWER 26 OF 162 LIFESCI COPYRIGHT 2008 CSA on STN DUPLICATE 13  
TI Levels of DNA damage are unaltered in mice overexpressing human catalase  
in nuclei  
SO Free Radical Biology & Medicine [Free Radical Biol. Med.], (20001001) vol.  
29, no. 7, pp. 664-673.  
ISSN: 0891-5849.  
AU Schriener, S.E.; Ogburn, C.E.; Smith, A.C.; Newcomb, T.G.; Ladiges, W.C.;  
Dolle, M.E.T.; Vijg, J.; Fukuchi, K.-I.; Martin, G.M.\*  
AN 2001:8012 LIFESCI

L85 ANSWER 27 OF 162 MEDLINE on STN DUPLICATE 14  
TI The peroxisomal targeting sequence type 1 receptor, Pex5p, and the  
peroxisomal import efficiency of alanine:glyoxylate aminotransferase.  
SO The Biochemical journal, (2000 Dec 1) Vol. 352 Pt 2, pp. 409-18.  
Journal code: 2984726R. ISSN: 0264-6021.  
AU Knott T G; Birdsey G M; Sinclair K E; Gallagher I M; Purdue P E; Danpure C  
J  
AN 2001087423 MEDLINE

L85 ANSWER 28 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
TI Molecular Mechanism of Detectable Catalase-Containing Particles,  
Peroxisomes, in Fibroblasts from a PEX2-Defective Patient  
SO Biochemical and Biophysical Research Communications (2000), 268(1), 31-35  
CODEN: BBRC9; ISSN: 0006-291X  
AU Shimozawa, Nobuyuki; Zhang, Zhongyi; Imamura, Atsushi; Suzuki, Yasuyuki;  
Fujiki, Yukio; Tsukamoto, Toshiro; Osumi, Takashi; Aubourg, Patrick;  
Wanders, Ronald J. A.; Kondo, Naomi  
AN 2000:74151 HCAPLUS  
DN 132:263642

L85 ANSWER 29 OF 162 MEDLINE on STN DUPLICATE 15  
TI Overexpression of wild-type and nuclear-targeted catalase modulates  
resistance to oxidative stress but does not alter spontaneous mutant  
frequencies at APRT.  
SO Mutation research, (2000 Apr 3) Vol. 449, No. 1-2, pp. 21-31.  
Journal code: 0400763. ISSN: 0027-5107.  
AU Schriener S E; Smith A C; Dang N H; Fukuchi K; Martin G M  
AN 2000217129 MEDLINE

L85 ANSWER 30 OF 162 LIFESCI COPYRIGHT 2008 CSA on STN DUPLICATE 16  
TI Localization of a Portion of Extranuclear ATM to Peroxisomes  
SO Journal of Biological Chemistry [J. Biol. Chem.], (19991100) vol. 274, no.  
48, pp. 34277-34282.  
ISSN: 0021-9258.  
AU Watters, D.; Kedar, P.; Spring, K.; Bjorkman, J.; Chen, P.; Gatei, M.;  
Birrell, G.; Garrone, B.; Srinivasa, P.; Crane, D.I.; Lavin, M.F.  
AN 2000:45813 LIFESCI

L85 ANSWER 31 OF 162 LIFESCI COPYRIGHT 2008 CSA on STN DUPLICATE 17  
TI Identification and Characterization of the Human Orthologue of Yeast  
Pex14p  
SO Molecular and Cellular Biology [Mol. Cell. Biol.], (19990300) vol. 19, no.  
3, pp. 2265-2277.  
ISSN: 0270-7306.  
AU Will, G.K.; Soukupova, M.; Hong, X.; Erdmann, K.S.; Kiel, J.A.K.W.; Dodt,  
G.; Kunau, W.; Erdmann, R. \*  
AN 1999:37867 LIFESCI

L85 ANSWER 32 OF 162 MEDLINE on STN DUPLICATE 18  
TI Oxidation of human catalase by singlet oxygen in myeloid

leukemia cells.

SO Photochemistry and photobiology, (1999 Dec) Vol. 70, No. 6, pp. 887-92.  
Journal code: 0376425. ISSN: 0031-8655.

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L85 ANSWER 87 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation  
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L85 ANSWER 143 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

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CODEN: ZNCBDA; ISSN: 0341-0382

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OREF 92:2891a,2894a

L85 ANSWER 144 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

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CODEN: BBGSB3; ISSN: 0304-4165

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L85 ANSWER 146 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

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L85 ANSWER 147 OF 162 MEDLINE on STN DUPLICATE 92

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L85 ANSWER 148 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 93

TI ON THE SYNTHESIS AND DEGRADATION OF THE MULTIPLE FORMS OF CATALASE  
EC-1.11.1.6 IN MOUSE LIVER EFFECTS OF AMINO TRIAZOLE AND P CHLOROPHENOXY  
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STN DUPLICATE 94

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L85 ANSWER 150 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
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CODEN: EXPTAX; ISSN: 0014-4908

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L85 ANSWER 152 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
STN DUPLICATE 96

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L85 ANSWER 153 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

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Journal code: 0411300. ISSN: 0301-5564.

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L85 ANSWER 155 OF 162 MEDLINE on STN DUPLICATE 98

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L85 ANSWER 159 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
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OREF 82:11079a,11082a

L85 ANSWER 160 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
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CODEN: JOBIAO; ISSN: 0021-924X

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OREF 79:2615a,2618a

L85 ANSWER 161 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
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L85 ANSWER 162 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN  
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L85 ANSWER 6 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on  
STN

L85 ANSWER 10 OF 162 MEDLINE on STN DUPLICATE 4  
AB In this study we cloned CTA1, the gene encoding peroxisomal catalase, from the methylotrophic yeast *Candida boidinii* and studied targeting of the gene product, Ctalp, into peroxisomes by using green fluorescent protein (GFP) fusion proteins. A strain from which CTA1 was deleted (ctalDelta strain) showed marked growth inhibition when it was grown on the peroxisome-inducing carbon sources methanol, oleate, and D-alanine, indicating that peroxisomal catalase plays an important nonspecific role in peroxisomal metabolism. Ctalp carries a peroxisomal targeting signal type 1 (PTS1) motif, -NKF, in its carboxyl terminus. Using GFP fusion proteins, we found that (i) Ctalp is transported to peroxisomes via its PTS1 motif, -NKF; (ii) peroxisomal localization is necessary for Ctalp to function physiologically; and (iii) Ctalp is bimodally distributed between the cytosol and peroxisomes in methanol-grown cells but is localized exclusively in peroxisomes in oleate- and D-alanine-grown cells. In contrast, the fusion protein GFP-AKL (GFP fused to another typical PTS1 sequence, -AKL), in the context of CbPmp20 and D-amino acid oxidase, was found to localize exclusively in peroxisomes. A yeast two-hybrid system analysis suggested that the low transport efficiency of the -NKF sequence is due to a level of interaction between the -NKF sequence and the PTS1 receptor that is lower than the level of interaction with the AKL sequence. Furthermore, GFP-CtalpDeltankf coexpressed with Ctalp was successfully localized in peroxisomes, suggesting that the oligomer was formed prior to peroxisome import and that it is not necessary for all four subunits to possess a PTS motif. Since the main physiological function of catalase is degradation of H<sub>2</sub>O<sub>2</sub>, suboptimal efficiency of catalase import may confer an evolutionary advantage. We suggest that the PTS1 sequence, which is found in peroxisomal catalases, has evolved in such a way as to give a higher priority for peroxisomal transport to peroxisomal enzymes other than to catalases (e.g., oxidases), which require a higher level of peroxisomal transport efficiency.

L85 ANSWER 14 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
STN

L85 ANSWER 20 OF 162 MEDLINE on STN DUPLICATE 9  
AB We established a Chinese hamster ovary cell line having a temperature-sensitive phenotype in peroxisome biogenesis. This mutant (65TS) was produced by transforming a PEX2-defective mutant, Z65, with a mutant PEX2 gene, PEX2(E55K), derived from a patient with infantile Refsum disease, a milder form of peroxisome biogenesis disorder. In 65TS,

catalase was found in the cytosol at a nonpermissive temperature (39 degrees C), but upon the shift to a permissive temperature (33 degrees C), catalase gradually localized to the structures containing a 70-kDa peroxisomal membrane protein, PMP70. In contrast to catalase, other matrix proteins containing typical peroxisome targeting signals, acyl-CoA oxidase and peroxisomal 3-ketoacyl-CoA thiolase, were co-localized with PMP70 in most cells, even at 39 degrees C. We found that these structures are partially functional peroxisomes and named them "catalase-less peroxisomes." Catalase-less peroxisomes were also observed in human fibroblasts from patients with milder forms of peroxisome biogenesis disorder, including the one from which the mutant PEX2 gene was derived. We suggest that these structures are the causes of the milder phenotypes of the patients. Temperature-dependent restoration of the peroxisomes in 65TS occurred even in the presence of cycloheximide, a protein synthesis inhibitor. Thus, we conclude that in 65TS, catalase-less peroxisomes are the direct precursors of peroxisomes.

L85 ANSWER 28 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

AB Patients with peroxisome biogenesis disorders (PBD) can be identified by detection of peroxisomes in their fibroblasts, by immunocytochem. staining using an anti-catalase antibody. The authors report here data on three PBD patients with newly identified mutations (del550C and del642G) in the PEX2 gene which encodes a 35-kDa peroxisomal membrane protein containing two membrane-spanning and a C-terminal cysteine-rich region. Some of the fibroblasts from the patient with the del642G mutation contained numerous catalase-containing particles, whereas no fibroblasts containing such particles were found in the patient with the del550C mutation. The authors confirmed that the del642G mutation caused a partial defect in peroxisome synthesis and import by expression of the mutated PEX2 into PEX2-defective CHO mutant cells. The authors propose that the two putative membrane-spanning segments in Pex2p are important domains for peroxisome assembly and import and that a defect in one of these domains severely affects PBD patients. Furthermore, a defect in the C-terminal portion of Pex2p exposed to the cytosol containing a RING finger motif caused the mild phenotype, residual enzyme activities, and mosaic detectable peroxisomes in fibroblasts from the patient. (c) 2000 Academic Press.

L85 ANSWER 38 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

AB Peroxisome proliferators are non-genotoxic hepatic carcinogens which may act by increasing oxidative damage in the liver. To test this hypothesis, we produced a transgenic mouse line with elevated catalase activity in the liver. We examined whether catalase overexpression influences the induction of lipid peroxidn. or oxidative DNA damage, two mechanisms deemed important in the carcinogenesis by peroxisome proliferators. Transgenic mice or non-transgenic litter mates were fed 0.01% ciprofibrate diet or a control diet for 21 days. The activities of fatty acyl CoA oxidase and lauric acid hydroxylase were not much affected by catalase overexpression, although the ratio of fatty acyl CoA oxidase to catalase activities was decreased in the transgenic animals. Hepatic lipid peroxidn. was estimated by the formation of malondialdehyde and conjugated dienes. Ciprofibrate did not affect either endpoint, but the catalase overexpression increased the concns. of malondialdehyde (in untreated mice only) and conjugated dienes (in both untreated and ciprofibrate-fed mice). Oxidative DNA damage was estimated by quantifying 8-hydroxydeoxyguanosine (8-OHdG) by HPLC with electrochem. detection. Ciprofibrate increased the hepatic 8-OHdG concns. but catalase overexpression did not affect them, although 8-OHdG concns. were decreased 50% in untreated mice. The metabolism of hydrogen peroxide by catalase may not be an important factor in the development of hepatic lipid peroxidn. The decrease in hepatic 8-OHdG in untreated transgenic mice and the increase after ciprofibrate administration imply that hydrogen peroxide is

important in the formation of 8-OHdG. While the lack of decreased 8-OHdG levels in ciprofibrate-treated transgenic mice does not support this conclusion, it is possible that catalase levels were not sufficiently high to affect this endpoint. Transgenic mice with higher hepatic catalase activities may be required to resolve this issue.

L85 ANSWER 50 OF 162 MEDLINE on STN DUPLICATE 28

AB Catalase is a ubiquitous peroxisomal matrix enzyme, yet the molecular targeting signal(s) for sorting it in plant cells has not been defined. The most common peroxisome targeting signal (PTS) is a C-terminal tripeptide composed of a conserved SKL motif (type 1 PTS). The PTS for cottonseed catalase (Ccat) was elucidated in this study from immunofluorescence microscopic analyses of tobacco BY-2 suspension cells serving as an in vivo import system. To distinguish biolistically introduced Ccat from endogenous tobacco catalase, Ccat was hemagglutinin (HA)epitope-tagged at its N-terminus. Bombardment with HA-Ccat resulted in the import of Ccat into glyoxysomes, the specialized type of peroxisome in BY-2 cells. The C-terminal tripeptide of Ccat, PSI, is necessary for import. Evidence for this were mislocalizations to the cytosol of PSI-truncated Ccat and AGV-substituted (for PSI) Ccat. PSI-COOH, however, was not sufficient to re-route chloramphenicol acetyltransferase (CAT) from the cytosol to glyoxysomes, whereas the Ccat tetrapeptide RPSI-COOH was sufficient. Surprisingly, substitution of K (common at the fourth position in other plant catalases) for the R (CAT-KPSI) decreased import efficiency. However, substitution of K did not affect import, when additional upstream residues in Ccat were included (e.g. CAT-NVKPSI). Other evidence for the importance of upstream residues comprised abolishment of Ccat import due to substitutions with non-conserved residues (e.g. -AGVNV RPSI for -SRLNV RPSI). These data indicate that Ccat is sorted to plant peroxisomes by a degenerate type 1 PTS (PSI-COOH) whose residues are functionally dependent on a strict context of adjacent C-terminal amino acid residues.

L85 ANSWER 51 OF 162 HCAPLUS COPYRIGHT 2008 ACS on STN

AB In healthy mice dietary treatment with 10% fish oil induced a significant and time-dependent increase in hepatic peroxisomal  $\beta$ -oxidation, catalase activity and peroxisomal number. In contrast a diet with 2% docosahexaenoic acid increased only the activity of catalase, suggesting that this polyunsatd. fatty acid is not the only inducing component in a high-fish oil diet. Myocardial catalase activity was induced both in mice fed diets with 10% fish oil and with 2% docosahexaenoic acid. Doses resembling therapeutic doses prescribed to patients with peroxisomal diseases slightly induced peroxisomal enzyme activities in liver and myocardium while peroxisomal morphol. remained unchanged. Renal changes were absent. Dietary treatment with several doses of Lorenzo's Oil had no effect on hepatic and renal peroxisomal parameters, while myocardial catalase activity was induced in most groups. The lack of hepatic peroxisomal changes in mice fed 20% Lorenzo's Oil strongly suggests that oleic acid and erucic acid are not the main inducers of the peroxisomal alterations observed in rodents treated with high-fat diets containing these fatty acids. The findings that doses of docosahexaenoic acid and Lorenzo's Oil corresponding to doses prescribed to peroxisomal patients, have little effect on hepatic peroxisomal enzymes in healthy mice, suggest that these diet supplements most probably will not add a strong load to the already disturbed fatty acid metabolism of the patients. The induction of myocardial catalase activity by therapeutic doses of docosahexaenoic acid and Lorenzo's Oil, requires addnl. studies.

L85 ANSWER 52 OF 162 MEDLINE on STN DUPLICATE 29

AB The peroxisomal disorders represent a group of inherited metabolic

disorders that derive from defects of peroxisomal biogenesis and/or from dysfunction of single or multiple peroxisomal enzymes. We described earlier an 8 1/2 year-old with a history of progressive developmental delay, micronodular cirrhosis, and elevated very long chain fatty acids in plasma and skin fibroblasts. These findings were felt to be compatible with both neonatal adrenoleukodystrophy (nALD) and Zellweger syndrome (ZS). This patient is now 21 years old and his clinical course, inconsistent with either nALD or ZS, led us to examine his peroxisomal status in light of a possible new peroxisomal disease. The normal levels of bile acid precursors found in this patient suggest that peroxisomal beta-oxidation is functional. The activities of dihydroxyacetone phosphate acyltransferase and oxidation of lignoceric acid and phytanic acid were 14, 17, and 15% of the control, respectively. This partial activity for oxidation and the normal levels of bile acid precursors suggests that this patient has peroxisomes containing beta-oxidation enzymes. Western blot analysis of subcellular organelles showed that beta-oxidation enzyme proteins are present at normal levels in catalase-negative peroxisomes of density equivalent to normal peroxisomes. The presence of acyl-CoA oxidase and 3-ketoacyl-CoA thiolase in catalase-negative peroxisomes suggests that both peroxisomal targeting signal-1 (PTS-1), and peroxisomal targeting signal-2 (PTS-2)-mediated protein transport processes into peroxisomes are normal in this patient. These findings of catalase-negative peroxisomes of normal density and normal PTS-1 and PTS-2 import machinery with partial peroxisomal functions clearly demonstrate that this patient differs from those with known disorders of peroxisomal biogenesis.

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L85 ANSWER 56 OF 162 MEDLINE on STN DUPLICATE 32

AB We have identified a novel peroxisomal targeting sequence (PTS) at the extreme COOH terminus of human catalase. The last four amino acids of this protein (-KANL) are necessary and sufficient to effect targeting to peroxisomes in both human fibroblasts and *Saccharomyces cerevisiae*, when appended to the COOH terminus of the reporter protein, chloramphenicol acetyl transferase. However, this PTS differs from the extensive family of COOH-terminal PTS tripeptides collectively termed PTS1 in two major aspects. First, the presence of the uncharged amino acid, asparagine, at the penultimate residue of the human catalase PTS is highly unusual, in that a basic residue at this position has been previously found to be a common and critical feature of PTS1 signals. Nonetheless, this asparagine residue appears to constitute an important component of the catalase PTS, in that replacement with aspartate abolished peroxisomal targeting (as did deletion of the COOH-terminal four residues). Second, the human catalase PTS comprises more than the COOH-terminal three amino acids, in that COOH-terminal-ANL cannot functionally replace the PTS1 signal-SKL in targeting a chloramphenicol acetyl transferase fusion protein to peroxisomes. The critical nature of the fourth residue from the COOH terminus of the catalase PTS (lysine) is emphasized by the fact that substitution of this residue with a variety of other amino acids abolished or reduced peroxisomal targeting. Targeting was not reduced when this lysine was replaced with arginine, suggesting that a basic amino acid at this position is required for maximal functional activity of this PTS. In spite of these unusual features, human catalase is sorted by the PTS1 pathway, both in yeast and human cells. Disruption of the *PAS10* gene encoding the *S. cerevisiae* PTS1 receptor resulted in a cytosolic location of chloramphenicol acetyl transferase appended with the human catalase PTS, as did expression of this protein in cells from a neonatal

adrenoleukodystrophy patient specifically defective in PTS1 import. Furthermore, through the use of the two-hybrid system, it was demonstrated that both the PAS10 gene product (Pas10p) and the human PTS1 receptor can interact with the COOH-terminal region of human catalase, but that this interaction is abolished by substitutions at the penultimate residue (asparagine-to- aspartate) and at the fourth residue from the COOH terminus (lysine-to-glycine) which abolish PTS functionality. We have found no evidence of additional targeting information elsewhere in the human catalase protein. An internal tripeptide (-SHL-, which conforms to the mammalian PTS1 consensus) located nine to eleven residues from the COOH terminus has been excluded as a functional PTS. Additionally, in contrast to the situation for *S. cerevisiae* catalase A, which contains an internal PTS in addition to a COOH-terminal PTS1, human catalase lacks such a redundant PTS, as evidenced by the exclusive cytosolic location of human catalase mutated in the COOH-terminal PTS. Consistent with this species difference, fusions between catalase A and human catalase which include the catalase A internal PTS are targeted, at least in part, to peroxisomes regardless of whether the COOH-terminal human catalase PTS is intact.

L85 ANSWER 58 OF 162 MEDLINE on STN DUPLICATE 34

L85 ANSWER 59 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN

L85 ANSWER 63 OF 162 MEDLINE on STN DUPLICATE 37

AB The molecular signal for targeting catalases to peroxisomes has not been defined. In this study, a plant in vivo import system (tobacco BY-2 suspension culture cells) was used to test the current postulate that the peroxisome targeting signal (PTS) for mammalian catalases is the internal Ser-Lys-Leu (SKL) motif found approximately eight amino acid residues from the C-terminus. Elucidation of the catalase PTS has been hampered previously by the ubiquitous presence of catalase in peroxisomes. The current study was possible because antibodies to mammalian catalases did not recognize endogenous, tobacco peroxisome catalase. Rat and mouse liver catalases (Rcat and Mcat), with an internal Ser-His-Ile (SHI) and Ser-His-Met (SHM), respectively, and both with a C-terminal Ala-Asn-Leu (ANL), were expressed transiently in BY-2 cells and targeted to the peroxisomes. Sorting was demonstrated by double-label immunofluorescence colocalization of these catalases with tobacco catalase. Peroxisome targeting of Rcat was abolished as expected when the internal SHI residues were removed by deletion of three C-terminal portions (28, 16, or 11 residues). Surprisingly, peroxisome targeting was still abolished when SHI (or SHL produced by site-directed mutagenesis) were at the extreme C-terminus as a consequence of deleting eight residues. However, when SHL was at the C-terminus in full-sized Rcat via a mutation of ANL-COOH, the enzyme sorted to peroxisomes indicating that the position of the PTS is significant in Rcat. The importance of the internal context of the SHI (or SHL) was examined further by changing ANL-COOH to a non-SKL motif, AGS-COOH. This Rcat did not sort to the peroxisomes, nor did Rcat with its ANL-COOH deleted; these data indicated the necessity of the C-terminal tripeptide. Sufficiency of ANL was demonstrated when chloramphenicol acetyltransferase with an appended ANL-COOH was redirected from the cytosol to peroxisomes. Collectively, these results do not support the internal PTS hypothesis, but indicate that a type 1 PTS slightly divergent from the typical SKL motif serves as the necessary and sufficient PTS for rat liver and probably other eukaryotic catalases.

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L85 ANSWER 74 OF 162 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation  
on STN DUPLICATE 43

L85 ANSWER 86 OF 162 MEDLINE on STN DUPLICATE 51

AB In contrast to many other peroxisomal proteins catalase A contains at least two peroxisomal targeting signals each sufficient to direct reporter proteins to peroxisomes. One of them resides at the extreme carboxy terminus constituting a new variant of this signal, -SSNSKF, not active in monkey kidney cells (Gould, S. J., G. A. Keller, N. Hosken, J. Wilkinson, and S. Subramani 1989. J. Cell Biol. 108:1657-1664). However, this signal is completely dispensable for import of catalase A itself. In its amino-terminal third this protein contains another peroxisomal targeting signal sufficient to direct reporter proteins into microbodies. This internal signal depends on the context. The nature of this targeting signal might be a short defined sequence or a structural feature recognized by import factors. In addition, we have demonstrated that the carboxy-terminal seven amino acids of citrate synthase of *Saccharomyces cerevisiae* encoded by CIT2 and containing the canonical -SKL represents a targeting signal sufficient to direct reporter proteins to peroxisomes.

L85 ANSWER 89 OF 162 MEDLINE on STN DUPLICATE 53

AB Hepatic peroxisomes in human embryos with a menstrual age of 6 and 7 weeks have been examined via catalase cytochemistry. In the younger sample, the organelles show no catalase activity, their matrix being pale and coarsely reticular. In the 7-week specimen, the peroxisome population consists of catalase-positive and catalase-negative organelles. The latter have a morphology identical to that of the 6-week sample and represent 66% of the population. The positive organelles show a pronounced staining heterogeneity. Together with the simultaneous presence of negative organelles, this might reflect the onset of catalase import into the peroxisomes during this period. Catalase heterogeneity excludes a continuous exchange of matrix contents; moreover, interconnections between peroxisomes have not been observed, and no cluster formation occurs. The data therefore also suggest that catalase is imported into individual, preexisting organelles in embryonic liver. The three peroxisomal beta-oxidation enzymes become detectable by immunocytochemistry only later during development. Morphological indications for a rapidly dividing population, such as elongated and/or tailed organelles, have not been observed. Morphometry has revealed that, in these early stages, the organelles are significantly smaller than the peroxisomes of fetal and adult human liver.

L85 ANSWER 90 OF 162 MEDLINE on STN DUPLICATE 54

AB To study the conservation of peroxisomal targeting signals, we have determined the intracellular localization of human peroxisomal catalase when expressed in yeast. Using immunofluorescence, differential centrifugation and immunoelectron microscopy, we show that the protein is targeted to the peroxisomes of the heterologous cell and assembled in its active tetrameric form. These data show the conservation of the catalase targeting signal and import specificity between human and yeast peroxisomes.

L85 ANSWER 91 OF 162 MEDLINE on STN DUPLICATE 55

AB The biogenesis, assembly and import of the peroxisomal enzyme catalase was studied in human skin fibroblasts from control persons and from patients with the Zellweger syndrome. For this purpose, two monoclonal antibodies were generated which are able to discriminate between the monomeric or dimeric form and the tetrameric, enzymically active conformation of the enzyme. Metabolic labelling studies showed that catalase is assembled to the tetrameric conformation

within one hour after its synthesis, while it is still in the cytosol of the cell. Subsequently, the enzyme becomes particle-bound in the control cells, a process that is retarded by addition of the catalase inhibitor 3-amino-1,2,4-triazole. However, the tetramer remains in the cytosol in cells from Zellweger patients. It is concluded that newly synthesized catalase can be assembled to a tetramer in the cytosol in human skin fibroblasts. Unfolding of this tetramer prior to import into peroxisomes is indicated.

- L85 ANSWER 92 OF 162 MEDLINE on STN DUPLICATE 56  
AB The minimal sequence requirement for a peroxisome-targeting signal was investigated using an in vitro import system. Carboxyl-terminal sequences Ser-Lys-Leu (SKL) and Leu-Gln-Ser-Lys-Leu (LQSKL) of acyl-CoA oxidase (AOX) directed to peroxisomes the fused proteins with import-incompetent forms of AOX and catalase that had been truncated, implying that the SKL tripeptide functions as a targeting signal. Elimination of the entire SKL sequence or deletion of any 1 or 2 amino acids in the sequence abolished the import activity of AOX. Substitution of alanine for serine did not affect the import activity. Topogenic activity was retained when lysine was mutated to either arginine or histidine, whereas mutation to glutamic acid completely abolished the activity. A synthetic peptide comprising the carboxyl-terminal 10 amino acid residues of AOX inhibited the import of the authentic AOX polypeptide, whereas other peptides in which SKL was mutated, deleted, or internally located were not effective. The uptake of AOX was little affected by the peptide with an amidated alpha-carboxyl group. These results strongly suggest that the carboxyl-terminal SKL motif sequence (Ser/Ala)-(Lys/Arg/His)-Leu functions as a topogenic signal in translocation of proteins into peroxisomes, requiring the whole tripeptide sequence with a free alpha-COOH group at the carboxyl terminus.
- L85 ANSWER 93 OF 162 MEDLINE on STN DUPLICATE 57  
AB Previous work has shown that the firefly (*Photinus pyralis*) luciferase contains a C-terminal peroxisomal targeting signal consisting of the tripeptide Ser-Lys-Leu. This report describes the microinjection of two proteins, (i) luciferase and (ii) albumin conjugated to a peptide ending in the sequence Ser-Lys-Leu, into mammalian cells grown in tissue culture. Following microinjection, incubation of the cells at 37 degrees C resulted in peroxisomal transport of these exogenous proteins into catalase-containing vesicles. The translocation was both time and temperature dependent. The transport could be inhibited by coinjection of synthetic peptides bearing various peroxisomal targeting signal motifs. These proteins could be transported into peroxisomes in normal human fibroblast cell lines but not in cell lines derived from patients with Zellweger syndrome. These results demonstrate that microinjection of peroxisomal proteins yields an authentic in vivo system with which to study peroxisomal transport. Furthermore, these results reveal that the process of peroxisomal transport does not involve irreversible modification of the protein, that artificial hybrid substrates can be transported and used as tools to study peroxisomal transport, and that the defect in Zellweger syndrome is indeed the inability to transport proteins containing the Ser-Lys-Leu targeting signal into the peroxisomal lumen.
- L85 ANSWER 96 OF 162 MEDLINE on STN DUPLICATE 60  
AB The methylotrophic yeast, *Hansenula polymorpha*, harbours a unique catalase (EC 1.11.1.6), which is essential for growth on methanol as a carbon source and is located in peroxisomes. Its corresponding gene has been cloned and the nucleotide sequence determined. The deduced amino acid sequence displayed the tripeptide serine-lysine-isoleucine at the extreme C-terminus, which is similar to sequences of other peroxisomal targeting signals. Exchange of the ultimate amino acid, isoleucine, of catalase for

serine revealed a cytosolic enzyme activity and a concomitant loss of peroxisome function. We concluded that the tripeptide is essential for targeting of catalase in H. polymorpha.

- L85 ANSWER 97 OF 162 BIOTECHNO COPYRIGHT 2008 Elsevier Science B.V. on STN  
AB By both histological and biochemical criteria, peroxisomes in patients with Zellweger syndrome appear to be absent or severely deficient. By using 15-30% (wt/vol) Nycodenz/sucrose gradients to study the subcellular localization of extraperoxisomal catalase activity, a commonly used marker for mature peroxisomes, we detected a single peak of activity in Zellweger syndrome fibroblasts at an equilibrium density of 1.13 g/cm<sup>sup.3</sup>, lower than the expected 1.17 g/cm<sup>sup.3</sup> of mature peroxisomes. Upon recentrifugation in either the original gradient or one with a higher salt concentration, essentially all catalase activity was recovered in fractions of the original densities. The activity of the catalase peak was further analyzed by a digitonin titration and filtration assay in combination with Triton X-100 treatment. The catalase activity passed through 0.1- $\mu$ m and 0.22- $\mu$ m but was retained on 0.025- $\mu$ m membrane filters (mean pore size). After treatment with Triton X- 100 nearly all catalase activity passed through the filters. The results from fractionation data, digitonin latency measurement, and the detergent effect on the filtration behavior suggest that catalase is not free in the cytosol of Zellweger syndrome fibroblasts as commonly thought but in particles (W- particles). Similar low-density catalase-containing particles, distinct from peroxisomes, are also found in normal fibroblasts. We found that L- $\alpha$ - hydroxyacid oxidase, another peroxisomal matrix enzyme, is also present in W- particles derived from normal and Zellweger syndrome fibroblasts. We speculate that the low-density catalase -containing W-particle may represent an immature or incomplete form of peroxisome distinct from previously described ' peroxisomal ghosts' in Zellweger syndrome fibroblasts.
- L85 ANSWER 104 OF 162 MEDLINE on STN DUPLICATE 66  
AB Fusion of complementary cell lines from patients with diseases of peroxisome biogenesis leads to peroxisome assembly in the heterokaryons and to uptake of cytosolic catalase by the newly assembled peroxisomes. Here we show that catalase import is inhibited by prior binding to catalase of the inhibitor 3-amino-1,2,4-triazole, which appears to retard unfolding of the protein.
- L85 ANSWER 109 OF 162 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
- L85 ANSWER 117 OF 162 MEDLINE on STN DUPLICATE 74  
AB A mutant of the methanol-utilizing yeast Hansenula polymorpha defective in catalase was isolated. It lacks the ability to grow on methanol as the sole source of carbon and energy due to a loss of peroxisomal function that is required for the dissimilation and assimilation of this substrate. Growth of the mutant on glucose or glycerol was not impaired. Transformation of mutant cells with the gene coding for catalase A from Saccharomyces cerevisiae [Cohen, G., Fessl, F., Traczyk, J., Rytka, J. & Ruis, H. (1985) Mol. Gen. Genet. 200, 74-79] conferred constitutive expression of catalase activity. When the gene was placed under control of the regulatory methanol oxidase promoter from H. polymorpha, high levels of activity subject to glucose repression were obtained. In both cases efficient targeting of catalase A to the heterologous peroxisomes and assembly into an active form could be demonstrated. Concomitantly, growth on methanol was restored in the transformed mutant. The results are in line with a high conservation of transport signals on peroxisomal proteins. Expression of a cytosolic

catalase in *H. polymorpha* did not confer the ability to grow on methanol. Therefore, proper localization of the catalase activity is a prerequisite for peroxisomal function.

L85 ANSWER 118 OF 162 MEDLINE on STN DUPLICATE 75

AB The interaction of mouse liver catalase with subcellular membranes was studied, and an ionic interaction with a variety of membranes, including those derived from the microsomes, was observed. The interaction with microsomal membranes was found to be abolished by pre-treatment of catalase with neuraminidase, indicating a functional significance for catalase-bound sialic acid. Catalase activity was found to be enhanced when bound to membranes, and evidence for a weak association of catalase with peroxisomal structure in mouse liver was also obtained. It is concluded that mouse liver catalase has a capacity to bind to a variety of subcellular membranes in vivo and that this interaction may be consistent with a general protective role for the enzyme, as well as being compatible with a model of peroxisomal biogenesis which involves the interaction of catalase with microsomal membranes.

L85 ANSWER 119 OF 162 MEDLINE on STN DUPLICATE 76

AB Previous studies from our laboratories have shown that carcinogenic peroxisome proliferators significantly increase the mRNA levels of peroxisomal beta-oxidation genes in the rat liver by enhancing the transcriptional activity. Because of a good correlation between the inducibility of peroxisome proliferation and carcinogenicity of this class of xenobiotics, we proposed that sustained induction of peroxisomal beta-oxidation system and the resultant oxidative stress form the basis for carcinogenesis. Since this concept implies that tumors should develop only in tissues which display maximal peroxisome proliferation, we have now assessed the degree to which catalase and the three beta-oxidation genes are expressed in liver and 12 extrahepatic tissues of adult rats fed for 2 weeks a diet containing 0.025% ciprofibrate (w/w), a peroxisome proliferator. In the ciprofibrate-treated rats, the levels of catalase mRNA increased to less than 2-fold in liver, kidney, intestine, and heart, but no change was detected in other tissues. The mRNA levels of the three genes of beta-oxidation system in the liver of adult rats treated with ciprofibrate increased greater than 20-fold. In contrast, in the kidney, small intestine, and heart the increases in the mRNA levels of all three beta-oxidation genes were small and varied from 2- to 4-fold following ciprofibrate treatment. Ciprofibrate did not significantly increase the levels of these mRNAs in the other nine tissues. These results correlated well with the levels of peroxisomal beta-oxidation activity, peroxisome volume density, and the immunologically quantified proteins in various tissues. These results provide evidence for the presence of beta-oxidation enzymes in peroxisomes of many tissues of rat and for tissue (cell)-specific differences in the inducibility of mRNAs of these beta-oxidation genes. The marked inducibility of beta-oxidation genes in liver and subsequent development of liver tumors support the hypothesis that tumors develop in tissues that show inducibility of peroxisome proliferation vis a vis beta-oxidation system following exposure to peroxisome proliferators.

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